REMARKS

The specification has been amended to provide for the priority claim. Claims 1-15 have been cancelled without prejudice, and claims 16-23 have been added. No new matter has been added by virtue of the new claims. For instance, support for the new claims appears e.g. at page 10, lines 21-20; page 12, lines 15-22; Figure 1; and the original claims of the application.

Claims 5-6 and 8-10 were objected to for informalities, namely being dependent on non-elected claims. It is believed the new claims render this formalities objection moot.

Claims 2, 5-10 and 15 were rejected under 35 U.S.C. 112, second paragraph for formalities-type issues with certain claim language.

While Applicants fully disagree with the rejection, it is also believed that at least many grounds of rejection have been obviated by the new claims.

For instance, the claims have more formalized antecedent basis.

Moreover, recitation of "the presence" and "the absence" does not contravene the formalistic protocol of antecedent basis. Those terms are commonly recited with the article of "the". SO

In view thereof, reconsideration and withdrawal of the rejection are requested.

Applicants' methods for screening acetyltransferase inhibitor or enhancer includes an *acetylated peptide substrate* that is detected using an *anti-acetylated peptide antibody*. As recited in independent claim 16, the anti-acetylated peptide antibody recognizes an acetylated form of the peptide substrate and does not particularly recognize the peptide substrate in unacetylated form.

Claims 2, 5-6, 9 and 15 were rejected under 35 U.S.C. 102 over Lill et al. (Nature paper). The rejection is traversed.

As the rejection is understood, the position is taken at least implicitly that p300/CBP is considered to be an acetyltransferase.

However, that position does not withstand scrutiny. Among other things, the Lill et al. document does not describe ~~any acetyltransferase~~ activity of p300/CBP. Further, the Lill et al. document does not disclose that binding of p300/CBP to p53 results in acetylation of p53.

Nor does Lill et al. disclose other various aspects of Applicants' invention such as a method that comprise *detecting* the amount of "acetylated peptide substrate" produced in the presence of the test compound using "an anti-acetylated peptide antibody" and *comparing* the amount detected in the presence of the test compound to that detected in its absence.

Accordingly, the rejection should be withdrawn. See, for instance, *In re Marshall*, 198 USPQ at 346 ("[r]ejections under 35 USC 102 are proper only when the claimed subject matter is identically disclosed or described in the prior art.").

Claims 2, 5 and 15 were rejected under 35 U.S.C. 102 over Scolnick et al. (Cancer Research paper). The rejection is traversed.

Contrary to the apparent assertions set forth in the Office Action, the Scholnick et al. document does **not** disclose that p53 is acetylated by p300 and CBP.

Rather, the document indicates that if p53 binds CBP, p53 might activate transcription by recruitment of CBP, leading to histone acetylation and the weakening of the interaction of chromosomal DNA with histones. See Scolnick et al. at page 3696, left column.

Further, Scolnick et al. reports measuring p53 transcriptional activity by Western blotting using monoclonal antibodies. Scholnick et al. clearly does **not** report detecting an amount of acetylated p53 using an anti-acetylated p53 antibody as recited in Applicants' claims.

Yet further, Scolnick et al. reports detecting the presence of a p53-p300/CBP complex (i.e. the binding of p53 to p300/CBP) and not the acetylated-53 (i.e. the acetylation of p53) as recited in Applicants' claims, by using antibody PAb421. That Pab421 recognizes the COOH terminus of p53, rather than anti-acetylated p53 antibodies as Applicants disclose and claim. See Scolnick at page 3694, right column, second full paragraph.

Therefore, the rejection should be withdrawn. See, *In re Marshall, supra.*

Claims 2, 5-6 and 15 were rejected under 35 U.S.C. 102 over Gu et al. (Cell paper). The rejection is traversed.

The Gu et al. document also is distinct. For instance, the Gu et al. document reports using radiolabelled [^{3}H] sodium acetate. The Gu et al. document does **not** disclose use of anti-acetylated p53 antibodies specific for the acetylated form of p53 as Applicants disclose and claim. See Gu et al. at page 596, right column through page 597, left column.

Accordingly, reconsideration and withdrawal of the rejection are requested. See, *In re Marshall, supra.*

Claims 2, 8 and 10 were rejected under 35 U.S.C. 102 over Poethke et al. (J. Neuroimmuno. 1997). The rejection is traversed.

Poethke et al. reports a certain method for detecting the mere presence of an enzyme per se. Nowhere does Poethke et al. mention detecting acetylated substrate using an anti-acetylated peptide antibody and the use of such a measurement to quantify the effect of a test compound on the enzyme's activity.

Accordingly, the rejection should be withdrawn.

Claim 7 was rejected under 35 U.S.C. 103 over Lill et al. (Nature paper) or Gu et al. (Cell paper). The rejection is traversed.

This rejection also is properly withdrawn. The combination of the cited documents fail to remedy the deficiencies of those documents as discussed above, e.g. the failure of the cited documents to suggest use of anti-acetylated p53 antibodies specific for the acetylated form of p53 as Applicants disclose and claim. Nor do either of the cited documents disclose use of biotin to label the peptide substrate. In view thereof, withdrawal of the rejection is requested.

It is believed the application is in condition for immediate allowance, which action is earnestly solicited.

Respectfully submitted,

Peter F. Corless (Reg. 33,860)
EDWARDS & ANGELL, LLP
Dike, Bronstein, Roberts & Cushman IP Group
P.O. Box 9169
Boston, MA 02209
Tel: 617-517-5557
Fax: 617-439-4170